Elsewhere in biology

A selection of interesting papers published last month in *Chemistry & Biology*'s sister journals, *Current Biology* and *Structure* with *Folding & Design*, chosen and summarized by the staff of *Chemistry & Biology*.

Chemistry & Biology August 1999, 6:R231–R233

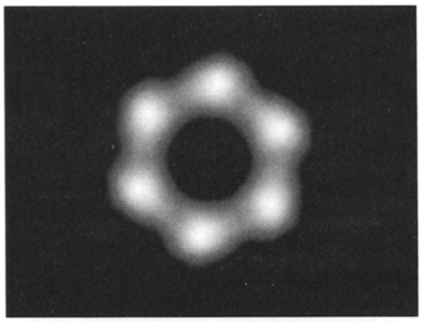
☐ **O-linked glycans mediate apical sorting of human intestinal sucrase-isomaltase through association with lipid rafts.**
Marwan Alfalah, Ralf Jacob, Ute Preuss, Klaus-Peter Zimmer, Hussein Naim and Hassan Y Naim (1999). *Curr. Biol.* **9**, 593–596.

The plasma membrane of polarised epithelial cells is characterised by two structurally and functionally different domains — the apical and basolateral domains. These domains contain distinct protein and lipid constituents that are sorted by specific signals to the correct surface domain. The best characterised apical sorting signal is that of glycophosphatidylinositol (GPI) membrane anchors, although N-linked glycans on some secreted proteins and O-linked glycans also function as apical sorting signals. The underlying sorting mechanisms for N-linked and O-linked

glycans remain obscure. Here, the authors have analysed the role of O-glycosylation in the apical sorting of sucrase-isomaltase (SI), a highly polarised N- and O-glycosylated intestinal enzyme, and the mechanisms underlying this process. The results demonstrate for the first time that O-linked glycans mediate apical sorting through association with lipid rafts.
24 May 1999, Brief Communication, *Current Biology*.

☐ **Oligomeric ring structure of the Bloom's syndrome helicase.**
Julia K Karow, Richard H Newman, Paul S Freemont and Ian D Hickson (1999). *Curr. Biol.* **9**, 597–600.

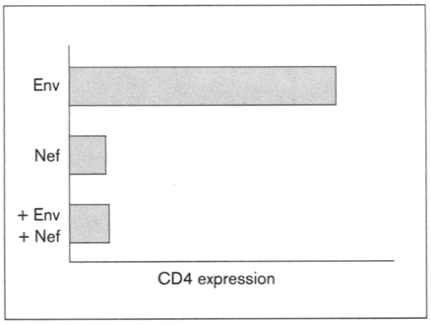
Bloom's syndrome is a recessive human genetic disorder associated with the early onset of many types of cancer, growth retardation and reduced fertility. The Bloom's syndrome gene product, BLM, belongs to the RecQ subfamily of DNA helicases and is required for the maintenance of genomic stability in human cells — in particular, the suppression of reciprocal exchanges



between sister chromatids. The authors have investigated the quaternary structure of BLM using a combination of size-exclusion chromatography and electron microscopy with reference-free image processing. They found that BLM forms hexameric ring structures surrounding a central hole. A fourfold symmetric square form that also had a central hole was also detected, which might represent a distinct oligomeric species or a side view of the hexameric form. Chromatography studies indicated that the majority of enzymatically active BLM has an apparent molecular mass of > 700 kDa, consistent with an oligomeric structure. These results have implications for the mechanism of action of BLM and suggest that other RecQ family helicases might also adopt ring structures.
24 May 1999, Brief Communication, *Current Biology*.

☐ **Inhibition of HIV-1 progeny virion release by cell-surface CD4 is relieved by expression of the viral Nef protein.**
Ted M Ross, Alp E Oran and Bryan R Cullen (1999). *Curr. Biol.* **9**, 613–621.

The HIV-1 Nef protein is required for efficient viral replication *in vivo* but has a number of distinct and apparently unrelated biological activities *in vitro*. One of these is the efficient internalization and degradation of cell-surface CD4, the receptor for the HIV-1 envelope protein, but the biological purpose of this internalization is unclear. Using human 293T cells expressing high levels of cell-surface CD4 or CD8, the authors demonstrate that CD4, but not CD8, can dramatically reduce the release of infectious virions bearing the HIV-1 envelope protein and induce a concomitant increase in the accumulation of cell-associated HIV-1 structural proteins. In contrast, CD4



had no effect on the release of HIV-1 bearing a heterologous envelope protein unable to bind CD4. *Nef* expression totally reversed CD4-mediated inhibition but only if the CD4 used remained susceptible to Nef-induced internalization. These results support the hypothesis that cell-surface CD4 can interact with the envelope protein present on budding HIV-1 virions to inhibit their release. The internalization and degradation of cell-surface CD4 induced by the viral Nef protein can fully reverse this inhibition and is, therefore, likely to facilitate the spread of virus *in vivo*.
27 May 1999, Research Paper, *Current Biology*.